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TO: HAPs Test Rule Reviewers

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FROM: Art Dungan (direct 202-872-4728) Art Dungan
(e-mail: adungan@c12.com)

DATE: September 29, 1997

SUBJECT: HAPs Comments
Record of Teleconference

Attached is a copy of the comments as submitted to EPA.

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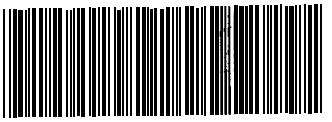

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September 29, 1997

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U.S. Environmental Protection Agency
Office of Pollution Prevention and Toxicities (OPPT)
Document Control Office (7407)
Rm. G-009
401 M Street, SW
Washington, D.C. 20460

Re: OPPTS-42 187A; FRL-4869- 1

Dear Sir/Madam:

The Chlorine Institute, Inc. is submitting the following comments concerning the Environmental Protection Agency's ***Proposed Test Rule for Hazardous Air Pollutants*** as presented in the Federal Register of June 26, 1996 and supplemented by several extensions for the comment period.

The Chlorine Institute (the Institute), founded in 1924, is a trade association of chlor-alkali manufacturers, users, repackagers, and suppliers to the industry. The Institute provides recommendations on safety, workers' health, and environmental protection regarding the production, distribution, and use of chlorine, sodium and potassium hydroxide, and sodium hypochlorite, and regarding the distribution and use of hydrogen chloride. The Institute's United States members are responsible for more than 98% of this country's production of chlorine, an annual figure of about thirteen million tons. The Institute has approximately 220 world wide members, of which approximately 75% are based in the United States.

SUMMARY

The Institute is providing comments for the proposed HAPs testing for hydrogen chloride and chlorine. The Institute believes that the EPA review of the existing toxicity studies for both hydrogen chloride and chlorine is incomplete.

The current information available for chlorine, when coupled with research underway are adequate for evaluation of effects on human health from exposure to chlorine. The agency may not be aware of activities currently being undertaken on chlorine by the Inhalation Toxicology Research Institute, the Chemical Industries Institute of Toxicology, and Dr. James Ultman of Pennsylvania State University. The results of these studies will be available to EPA upon their completion. The available and planned studies provide sufficient information to assess the relationship between exposure and the incidence and severity of portal of entry effects.

The Institute believes that currently there is adequate information for evaluation of effects on human health from exposure to hydrogen chloride and that obtaining additional information for hydrogen chloride as proposed in the June 26, 1996 Federal Register would not help in the risk assessment for this material nor would it provide any assistance in considering risk management options. Similarly the Institute believes that obtaining additional information for chlorine as proposed in this same Federal Register would not help in the risk assessment for this material nor would it provide any assistance in considering risk management options. The Institute believes that the information generated in the above mentioned CIIT - Ultman studies, when coupled with currently available information, will be adequate for health hazard assessment for acute exposure to chlorine.

The Institute believes that the EPA should eliminate any tests for hydrogen chloride when it promulgates the final HAPs rule and that EPA should eliminate the proposed HAPs tests for chlorine in the final rule.

The following specific comments regarding the proposed testing requirements for HCl and Cl, are presented.

HYDROGEN CHLORINE (HCl)

In the June 26 Federal Register the EPA proposed that acute inhalation studies be conducted with HCl under TSCA Section 4. As one of its key supporting documents the Agency lists TSCA SECTION 4 FINDINGS FOR 21 HAZARDOUS AIR POLLUTANTS *A Supporting Document for Proposed Hazardous Air Pollutants (HAPS) Test Rule.* This document concludes, for HCl, that "---there are inadequate data and experience to determine or predict the effects on human health---". We believe this conclusion is flawed for the reasons discussed below.

- **EPA has reviewed only a fraction of the existing acute toxicity studies with HCl.**

The support documentation briefly describes only two of the many acute inhalation studies that have been conducted with HCl. Hinderer and Kaplan (1986) provide an assessment of the inhalation toxicity of HCl to humans. In this document, the authors review in particular acute inhalation toxicity data on rodents, non-human primates, and humans dating from 1886 to 1984. More recent acute studies include Kaplan et al. (1993a), Kaplan et al. (1993b), and Kaplan et al. (1993c). We believe these studies are critical to providing a fuller understanding of the acute inhalation effects of HCl exposure.

- **EPA has not demonstrated that the existing data on HCI are inadequate.**

Since EPA failed to include the studies mentioned in the preceding section in its review, we feel it is inappropriate for the Agency to conclude that existing data on HCl are inadequate. All these studies are acute inhalation studies and collectively cover a range of exposure levels, duration of exposure, observation time, endpoints, and species differences. Additionally, some of the acute studies include histopathological examination of the respiratory tract. A review of these studies reveals that the rat and baboon are the best predictive species for human health effects. The studies document in detail minimal functional and morphological effects at exposure levels up to 4,200 ppm (rat) and 10,000 ppm (baboon) for exposure duration of 15 minutes and observation periods up to 360 days. In another study, Kaplan et al. (1984) investigated 5 minute exposure at 30,000 ppm and 10 minute exposure at 15,000 ppm in baboons. These studies provide adequate qualitative descriptions to predict the potential health effects of human exposure to HCl via inhalation. Based on the available information and data, predictions for human health effects can be made in any likely accidental scenario (as described in the following pages). Furthermore, we believe these studies were conducted under exposure conditions well in excess of those anticipated for humans in any likely accidental scenario.

In conclusion, these studies provide the best possible information using currently available techniques and methodology for determination of inhalation toxicity of chemicals. Additional generation of data will not provide any improvement in human health protection. However, if after reviewing the above mentioned studies, EPA arrives at a different conclusion, it should clearly demonstrate the inadequacy of the existing data and how any new information would provide better human health protection.

- **EPA fails to note that the rat is the most appropriate rodent model of the acute pulmonary damage or lethality of HCl in humans and that the mouse is not a good model.**

In assessing the predictive value of rodent data or in selecting a test species it is important that the Agency keep in mind that anatomical and physiological differences between rodents and higher mammals can be quite significant (McLaughlin et al., 1996; Erickson, 1989; Leith, 1984; Phalen, 1974; Avtandilov and Batsura, 1974). These species differences frequently make cross-species extrapolation difficult (Schlesinger, 1985). Such species differences can affect both the qualitative and quantitative responses that are observed.

A number of anatomical and physiological differences between rodents and primates can play a major role in the inhalation toxicity of a chemical. Rodents are obligate nasal breathers. They breathe only through their nose, while primates breathe through both their nose and mouth. Compared with primates, rodents have a more truncated respiratory tract with less branching and bronchiole alveolarization (Leith, 1984; McLaughlin et al., 1966). For chemicals such as HCl which are highly water soluble and rapidly react on surfaces, these differences can greatly influence deep lung penetration and the ability to produce significant histopathological findings. Furthermore, although both rodent and primate species exhibit a bronchoconstriction response when exposed to chemicals like methacholine (Allott et al., 1980; Chadra et al., 1984; Roehrs et al., 1981), rodents have completely different respiratory responses to simple irritants than primates. When rodents and primates are exposed to an irritant, rodent respiratory rate decreases while primate (non-human and human) respiratory rate increases (Kaplan et al., 1988; Amdur et al., 1952; Jiang et al., 1983; Burleigh-Flayer et al., 1985). Because metabolic rate is 7 times higher in mice and 4 times higher in guinea pigs and rats than man, decreased respiration may have a significant impact on the vital status of rodents.

Acute inhalation studies by Kaplan et al. (1985), Kaplan et al. (1988), Kaplan et al. (1993a), and Kaplan et al. (1993c) have shown that the rat is the best rodent model of the acute irritant effects of HCl in man.

In studies sponsored by the Federal Aviation Administration to assess human escape performance (Kaplan et al., 1985) rats and baboons were exposed for 5 minutes to levels as high as 87,660 ppm and 17,290 ppm, respectively, and then were observed for their ability to perform an operant behavioral task that enabled them to exit the exposure chamber. No evidence of incapacitation or decrease in escape performance was observed in either rats or baboons and the lowest lethal doses were found to be similar (16,570-30,000 ppm)[1]. In subsequent studies by Kaplan et al. (1988), Kaplan et al. (1993a), and Kaplan et al. (1993c) mice, guinea pigs, rats and baboons were exposed for 15 minutes to levels as high as 2,500 ppm, 4,200 ppm, 4,200 ppm, and 10,000 ppm, respectively, in order to compare the effects of HCl on pulmonary function and histopathology.

'Two baboons exposed to 16,570 ppm and 17290 ppm developed pulmonary infections and died 18 and 76 days post exposure (Kaplan et al., 1985); one baboon exposed to 30,000 ppm survived following a 5 minute exposure (Kaplan, 1984).

Again the response of the rat was found to be similar to that of the baboon. No deaths were observed in rats or baboons at levels as high as 4200 ppm and 10,000 ppm, respectively, with only minimal histopathological changes being evident at termination. In mice and guinea pigs deaths were observed at levels as low as 475 ppm and 520 ppm, respectively. Furthermore, severe damage to the respiratory tract, including pulmonary edema in some animals, was observed in those animals which died.

- **The existing data demonstrate that the upper respiratory tract is the primary target of HCl and that humans can survive high concentrations.**

HCl is a highly water soluble, reactive vapor. The major portals of entry for HCl in humans are expected to be the nose, or upper respiratory tract (URT), and mouth. Morris and Smith (1982) have shown that inspired hydrogen fluoride deposits in the nasal cavity of rats with greater than 99.7% efficiency, and is expected to be similar to other halide acids such as HCl. The high URT deposition efficiency and the physicochemical properties of highly water soluble and reactive compounds, like HCl, are expected to be Category 1 vapors as described by the U.S. EPA (1994). For Category I materials, dose is an important determinant of regional response patterns. Regional dose is dependent on airflow patterns which will vary between rodents and humans because of anatomical differences.

Deposition of vapors in the URT is controlled by resistance to gas phase mass transport, i.e. resistance to air phase molecular diffusion, solubility and reactivity in the mucus and tissue, metabolism, and blood flow removal from the tissue (Hanna et al., 1989; Morris, 1990).

Since HCl dissociates completely in the aqueous milieu of tissues, and is not metabolized, blood flow and metabolic clearance of HCl are not expected to be significant factors controlling deposition. Thus, mass transport of HCl to the tissue surface, i.e. delivered dose, and factors affecting its transport are likely to be the key determinants of 1) regional lesion distribution in the URT of rats, 2) concentration and exposure duration-dependent distribution of lesions, and 3) interspecies extrapolation of the dosimetry of HCl in the URT from rodents to humans.

The significant toxicological effects of HCl exposure are manifest at the site of contact. Thus, by the inhalation route, significant deposition is predicted to occur in the most anterior regions of the nasal cavity and extending posteriorly to the lower respiratory tract if sufficient exposure concentrations are achieved. After three, 6 hr/day exposures of mice to an HCl concentration of about 300 ppm, pathologic injury was limited almost exclusively to the respiratory epithelium (Buckley, et al., 1984). Histologically, the lesions induced at the sites of contact with HCl were seen in the trachea or lungs. Some recovery of the inflammation was observed at 72 hrs post-exposure, and was characterized by squamous metaplasia. However, complete recovery was not evident.

Recent work in the laboratories of Kimbell et al. (1993) and Hahn et al. (1993) has endeavored to develop computational models of airflow patterns in the rat and human URT. These models have been constructed using finite element modeling techniques such that three dimensional computational fluid dynamics (CFD) simulations of inspiratory airflow can be estimated in rats and humans. These models describe the major airflow patterns through the rat and human nose, taking into account the unique anatomical features of the two species.
For example, the rat nasal cavity is elongated. In the rat, air flows in streams that pass over well developed and complicated turbinates with a narrow passageway and small distances between the center of the air stream and the tissue surface. This arrangement presents a highly effective surface area for vapor deposition. The human nasal cavity, in contrast, is more spherical in shape with less complicated nasal turbinates yielding greater distances from the center of the air stream to the tissue lining. Airflow predictions from these models, visualized using three dimensional reconstruction of the nasal cavity surfaces, have been validated against experimental measures of water/dye streams or anemometric measures in models (Kimbell et al., 1993; Hahn et al., 1993)

The rat nasal model has recently been used to predict regions of high flux of formaldehyde vapor to the mucosal surface. Like HCl, formaldehyde is a highly reactive water soluble gas whose deposition and regional pattern of lesion formation is characteristic of a Category 1 gas.

Flux is defined as the rate of movement of mass from the air stream to the surface of the nasal cavity (units of mass/unit time/surface area). Like those studies with HCl, studies with formaldehyde show the high flux regions correlated well with regions of squamous metaplasia and inflammation, the toxic response of the nasal epithelium to formaldehyde gas. Thus, the regional flux corresponding to the NOAEL for high impact sites can be estimated and related to the inspired concentration. In this manner, an exposure level for humans that yield flux values equivalent to the NOAEL flux value in animals can be derived.

Because of concerns about species differences in response to HCl, the baboon has been used by investigators as a surrogate for man and to evaluate the ability of various rodent species to model or predict human response. This primate has been selected because of its anatomical and physiological similarities and parallelism to man (Collins and Jones, 1978). Furthermore, it has been used as a role model for human respiratory disease (Phalen, 1994).

The studies by Kaplan et al. (1988), Kaplan et al. (1993a), and Kaplan et al. (1993c) showed that the upper respiratory tract is the prime target of acute high level exposure to HCl. The exposure of groups of primates to 500, 5,000, or 10,000 ppm of HCl for 1.5 minutes resulted in decreased partial pressure of arterial oxygen (PaO_2) in the middle and high exposure groups.

Measurements using a pulmonary arterial (Swan-Ganz) catheter indicated that this was due to uneven ventilation resulting from bronchoconstriction of the conducting airways of the upper respiratory tract. Clinical observations and chest x-rays at one hour post-exposure and pulmonary function test results at 3 days, 3 months, and 6 months post-exposure did not reveal any evidence of damage to the lower respiratory tract. Lung function and histopathological changes occurred only in the 10,000 ppm group at 1 year post exposure, not in the 500 ppm or 5,000 ppm groups.

Although acute exposure to high concentrations of HCl can cause severe damage to the upper respiratory tract, the primate studies have indicated that man can survive high exposures. No mortalities have been observed in primates exposed to levels as high as 10,000 ppm for 15 minutes. Furthermore, Kaplan (1984)) has reported that baboons have survived HCl exposures of 30,000 ppm for 5 minutes and 5,000 ppm for 30 minutes.

- **Recommendation for Hydrogen Chloride**

The Institute believes there is currently adequate information available for evaluation of effects on human health from acute exposure to hydrogen chloride. Obtaining additional information for hydrogen chloride would not help in the risk assessment for this material nor would it provide any assistance in considering various risk management options.

The Institute believes the agency should eliminate any and all tests for hydrogen chloride when it promulgates its final rule for additional testing for the 21 hazardous air pollutants as proposed on June 26, 1996.

CHLORINE

In the June 26 Federal Register, the EPA proposed that acute inhalation studies be conducted with Chlorine under TSCA Section 4. In "TSCA SECTION 4 FINDINGS FOR 21 HAZARDOUS AIR POLLUTANTS", the EPA concludes that "the testing of chlorine is necessary to develop data for acute respiratory effects". A very substantial amount of toxicology information is available on chlorine, with assessment in a number of species, by acute through chronic exposure, and with a variety of toxicological endpoints. Additional acute inhalation toxicology studies are not needed, and will not usefully increase our knowledge of chlorine toxicity. The testing guideline that is proposed ("Acute inhalation toxicity with histopathology", OPPTS 870.1350) is complex with mandatory triggered testing that could result in an expensive and wasteful study. A relevant chlorine research program is ongoing at the Chemical Industry Institute of Toxicology (CIIT), and additional relevant studies are being conducted by the Chlorine Institute in conjunction with CIIT investigators and Dr. James Ultman of Penn State University.

- **EPA's review of relevant toxicology studies on chlorine is incomplete.**

Pulmonary toxicity, with histopathology, has been recently reported for rats exposed briefly to chlorine (Demnati et al, 1995). A mouse sensory irritation study is specified by the HAPS rule, but this information is already available, as reported by Barrow et al. (1977) and by Gagnaire et al. (1994).

Jiang et al. (1983) was cited by EPA's review, but was considered inadequate as only one dose level was used. Jiang et al. used light and scanning electron microscopy to characterize nasal lesions at what was considered to be the most relevant concentration for their study. This extensive data base on chlorine toxicology is fully adequate to assess exposure-response and establish safe exposure levels for acute exposure to chlorine. Additional acute testing is not warranted.

- **Existing data indicate that irritation of the respiratory tract is the primary adverse effect from inhalation exposure of chlorine.**

Excessive exposure to chlorine is irritating to the lungs, eyes, nose, and throat. Exposure to 1000 ppm killed mice in 28 minutes and rats in 53 minutes (Barrow et al. 1979). Another source reported a one-hour LC50 of 137 ppm for mice and 293 ppm for rats (Vernot et al. 1977). The LC50 for dogs (30 min exposure) was 650 ppm (Underhill, 1920). Some symptoms observed were fatal pulmonary edema, labored-respiration, and marked lacrimation.

Zwart and Woutersen (1988) reported on the acute inhalation toxicity of chlorine in rats and mice, providing detailed time-concentration-mortality assessments and histopathology. Satellite groups were sacrificed two days post-exposure for histopathology assessment, main groups were held for 14 days to assess survival. Edema, aggregates of inflammatory cells, hyperplasia of the lining epithelium of the larynx and trachea were among the histopathologic observations in rats after specified exposure regimens. The authors indicated that the estimated LC01 values (e.g. 3000 mg/m^3, 1000 ppm, for the 10 min LC01) seemed to correspond with long-term lung damage. Demnati et al. (1995) evaluated rats exposed to 50 to 1500 ppm chlorine for exposures of 2 to 10 minutes. Lower concentrations (500 ppm) did not induce significant histological changes. Exposure to 1500 ppm for 10 minutes caused substantial histopathological change - the course included edema (1 hour), appearance of mucosal polymorphonuclear lymphocytes (6 to 24 hours), epithelial cell regeneration (72 hours). A biochemical characterization of lungs was described by Dodd et al. (1980); lung sulfhydrol changes were measured following 1, 5, or 10 day inhalation exposure of rats.

The RD50 for mice is in the range of 3.5 ppm (Gagnaire et al., 1994) to 9.3 ppm (Barrow et al., 1977). Barrow et al. subsequently reported the development of sensory irritation tolerance in rats exposed to 0, 1, 5, or 10 ppm for two weeks prior to assessment of respiratory rate depression (1982).

Jiang et al. (1983) described the pathology of toxic responses to the RD50 concentration of chlorine gas in the nasal passages of rats and mice. Degeneration of olfactory sensory cells, particularly in the anterior portion of the dorsal meatus was described.

Lesions in the respiratory epithelium were located primarily in the free margins of the naso- and maxilloturbinates and adjacent nasal septum. Scanning electron microscopy revealed loss of cilia from the olfactory and respiratory epithelia.

A study which monitored the respiratory effects/function of persons acutely exposed to chlorine gas pursuant to a train derailment for 6 years after the incident suggests no persisting abnormal rate of decline in lung function as a result of the acute exposure (Jones et al, 1986). This 1978 accident was severe, eight deaths were reported by the authors, and 23 persons were hospitalized. Sixty adults were followed in the study, including 20 of the 23 hospitalized persons.
Changes in lung function correlated with smoking but not to distance from the exposure site or severity of injury suffered from exposure. There was no evidence of a persisting abnormal rate of pulmonary decline.

Construction workers, who remodeled a pulp mill over a 3 to 6 month time period, were followed for 18-24 months (Courteau et al., 1994; Bherer, et al. 1994).

The air monitoring data from the pulp mill industrial hygienist were reportedly not useful in linking specific events reported by the workers to environmental conditions in the bleach plant. Most of the workers had respiratory symptoms at the end of the follow up period. When follow up data are available, most workers or accident victims who survive a single exposure to substantial amounts appear to recover completely and relatively rapidly, though some individuals reportedly have hyper responsive airways over a protracted period of time.

Rotman et al. (1983) evaluated human volunteers exposed to low concentrations of chlorine. Subjects were initially exposed for a four hour period, followed by testing, then a second four hour exposure period. The testing scheme involved repeating these two 4-hour exposures providing two exposure days and one sham exposure day. Subjects engaged in exercise equivalent to light to moderate work for 15-minute intervals during exposure. Rotman et al. reported transient changes in pulmonary function measurements of volunteers exposed to 1 ppm; changes at 0.5 ppm were considered “trivial”.

Kusch et al. (1994) reported a prospective epidemiology study on manufacturing workers exposed to an average concentration of 0.092 ppm chlorine. There were no detectable effects on pulmonary function in these workers over the five year study.

Groups of 10 male and 10 female rats were exposed to 0 (controls), 1, 3, or 9 ppm chlorine for 6 hrs/day, 5 days/week for 6 weeks (30 exposures). Pulmonary effects were seen at all levels of exposure, and liver and kidney effects were noted at 3 and 9 ppm. Three females on the high dose died during the experiment. Urinary specific gravity, alkaline phosphatase, BUN, gamma-glutamyltranspeptidase, SGPT, hematocrit, and white blood cell counts were all elevated (Barrow et al., 1979).

Monkeys were exposed 6 hr./day, 5 days/week, for 12 months to 0, 0.1, 0.5, or 2.3 ppm (Klonne et al., 1987). Monkeys exposed to 2.3 ppm showed eye irritation but no cornea1 effects. Microscopic exam showed trace to mild metaplasia of the respiratory mucosal epithelium of the nasal cavity, and trace metaplasia of comparable cells in the trachea. Trace metaplasia of the respiratory mucosal epithelium of the nasal cavity was observed in some monkeys at lower concentrations, though the authors considered the changes below 2.3 ppm to be of questionable clinical significance.

In a chronic inhalation study by Wolf et al., male and female mice and rats were exposed to 0, 0.4, 1 .O, or 2.5 ppm chlorine for 6 hrs/day, 5 days/week for 2 years (1995). Female rats were exposed to the same concentration on 3 alternate days/week for 2 years. Exposure-dependent lesions were confined to the nasal passages in all sex and species groups.

However, there were no exposure-related neoplasms. Researchers concluded that airborne chlorine is an upper respiratory tract irritant but is not carcinogenic to mice or rats.

The repeated exposure studies done by Barrow et al. (1979), Klonne et al. (1987), and Wolf et al. (1995) describe histopathological effects on the respiratory tract. These studies were conducted at concentrations only slightly below the reported rat RD50 (3.5 to 9.3 ppm). Single exposure studies with histopathology would likely be close to this concentration range, and therefore would not be useful unless designed into a relevant research protocol.

The ACGIH reviewed inhalation data in 1991, including human studies, and recommended 0.5 ppm TWA, 1 ppm STEL as an exposure guideline to prevent eye and mucous membrane irritation from exposure to chlorine. The documentation provides a useful review. There are also substantial toxicology data by routes other than inhalation. Many of these were intended to relate to the use of chlorine as a water disinfectant. These are not reviewed here because the primary focus of this summary is inhalation toxicology.

A chlorine research program is underway (Ibanes,1996), and the following rat and human dosimetry studies are planned. These studies will be more useful for understanding the biological effects of chlorine, and particularly portal of entry effects, than additional acute toxicity tests.

Nasal dosimetry in animals: Includes measurement of chlorine uptake in surgically isolated trachea by CIIT investigators. Methods are based on those used for formaldehyde (Patterson et al., 1986). Dr. Alan Dahl of the Inhalation Toxicology Research Institute, Albuquerque, N.M., will be determining chlorine uptake in Rhesus monkeys (funded by EPA). Nasal uptake data will be used for dosimetry modeling of chlorine uptake patterns in rats, Rhesus monkeys, and humans. Initial work on rhesus monkeys (determination of the aifflow field) has been submitted for publication (Kepler et al., 1996).

CIIT investigators plan to examine nasal inflammation (rhinitis) mechanisms including the role of eosinophils and other inflammatory cells in the nose of rats.

Dosimetry patterns in humans are currently being studied by Dr. Ultman of Penn State University, in conjunction with CIIT investigators. Ultman plans to noninvasively determine the longitudinal distribution of chlorine uptake in the human respiratory tract. These methods were previously developed and applied to ozone by Ultman and co-workers (Hu et al., 1992). Integrals of the inhaled and exhaled chlorine concentration curves are used to determine the absorbed fraction and penetration (mean airway volume) reached by an inhaled Cl, bolus. The Chlorine Institute is sponsoring Ultman's research.

- **Recommendation for Chlorine**

The Institute believes the current information available for chlorine, when coupled with the research activities as described above and currently being undertaken are adequate for evaluation of effects on human health from exposure to chlorine. The Institute recommends that EPA defer any testing requirements until the on going work discussed above has been completed and the subsequent need for additional work has been evaluated. Obtaining additional information for chlorine would not help in the risk assessment for this chemical nor would it provide any assistance in considering various risk management options.

The Institute believes the agency should eliminate the tests proposed for chlorine when it promulgates its final rule for additional testing for the 21 hazardous air pollutants as proposed on June 26, 1996.

The Institute is most pleased to have an opportunity to provide these comments. Should additional information be needed, please contact Mr. Arthur E. Dungan, Vice President - Safety, Health, and Environment (direct phone line 202-872-4730).

Sincerely yours,

Robert G. Smerko

Robert G. Smerko

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